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Research report

Anticipation of a psychosocial stressor differentially influences ghrelin, cortisol and food intake among emotional and non-emotional eaters ☆

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ABSTRACT

Negative emotions trigger eating in some individuals (emotional eaters) possibly by influencing stress hormones that contribute to eating regulation (e.g., cortisol), or eating-related peptides (e.g., ghrelin) signaling food initiation. The present study assessed whether stressor-elicited cortisol and ghrelin changes would differ between emotional and non-emotional eaters, and whether eating would influence these neuroendocrine responses. Undergraduate women ($N = 103$) who completed measures of emotional eating, were assigned to anticipate either a stressful (public speaking) or non-stressful event. During this period, participants were or were not offered food. Blood samples were taken continuously over a 40-min period to assess changes of cortisol and ghrelin levels, and mood was assessed after the anticipation period. Baseline ghrelin levels were lower in emotional than non-emotional eaters, and this relation was mediated by percent body fat. Ghrelin levels were elevated among women anticipating a stressor, compared to those in the control condition. Additionally, the normal decline of ghrelin following food consumption was not apparent among emotional eaters. Although food intake was not tied to hormone responses, reported hunger was associated with greater food intake for women in the stressor condition. It was suggested that emotional eating coupled with subjective feelings of hunger, might contribute to eating in response to an acute stressor. Additionally, feedback mechanisms controlling the normalization of ghrelin levels might be disturbed in emotional eaters. The similarity of the ghrelin profile of emotional eaters to that of binge eaters and obese individuals, raises the possibility that disturbed ghrelin response might be a risk factor for such conditions.

Introduction

Stressful events may either reduce or increase eating, depending on the nature and/or severity of the stressor (Lattimore & Caswell, 2004; Newman, O'Connor & Conner, 2007; Stone & Brownell, 1994), and whether the individual is prone to being an emotional eater (van Strien, 2010; van Strien, Frijters, Bergers, & Defares, 1986), possibly reflecting a form of affect-regulation (Corsica & Spring, 2008; Stice, Spoor, Bohon, Veldhuizen, & Small, 2008). Studies in rodents likewise indicated that although stressors typically elicit reduced eating, under some conditions (e.g., chronic stress) stressful events may provoke elevated food consumption (Dallman et al., 2003), particularly in the form of highly palatable foods (Dallman, Pecoraro, & la Fleur, 2005; Kandiah, Yake, Jones, & Meyer, 2006; Zellner et al., 2006).

The release of stress-hormones, such as corticosterone, might contribute to increased consumption of palatable foods (Dallman et al., 2005; Epel, Lapidus, McEwen, & Brownell, 2001). This could occur either through the increased salience of the pleasurable effects of eating owing to glucocorticoid interactions with the dopamine reward processes (Adam & Epel, 2007; Berthoud & Morrison, 2008), by glucocorticoid stimulation of endocannabinoid receptors and other target neurons of the hypothalamus (Dallman, 2007; Di, Malcher-Lopes, Halmos, & Tasker, 2003), or by glucocorticoid inhibition of the anorectic effects of corticotropin releasing hormone (CRH) within the paraventricular nucleus of the hypothalamus (PVN) (Laugero, Gomez, Manalo, & Dallman, 2002; Nieuwenhuizen & Rutters, 2008). It is also possible that in a distressing context, food consumption that is interpreted as being positive, would serve as a form of self-medication or a means of coping with the stressful situation (Tomiyama, Dallman, & Epel, 2011).

In addition to a role for glucocorticoid and peptide factors that have been implicated in eating processes, ghrelin is known for its

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involvement in the regulation of eating, having been associated with increased feelings of hunger, eating initiation, as well as increased caloric intake (Cummings, Frayo, Marmonier, Aubert, & Chapelot, 2004; Schmid et al., 2005; Wren et al., 2001). Furthermore, ghrelin levels fluctuate over the day, peaking before meals and during fasting (Cummings et al., 2001; Tschöp, Smiley, & Heiman, 2000), and decreasing shortly after food consumption (Aydin et al., 2006; Cummings et al., 2002, 2004). Beyond the influence of ghrelin in normal eating patterns, altered ghrelin patterns have been observed among women presenting with clinical levels of eating disorders (Geliebter et al., 2004; Monteleone et al., 2000). Moreover, alterations of ghrelin patterns have been observed among women with sub-clinical eating disturbances such as emotional eating. Specifically, emotional eaters demonstrated lower baseline levels of ghrelin than non-emotional eaters and did not display the expected decrease of ghrelin typically observed after food intake and observed among non-emotional eaters (Raspopow, Abizaid, Matheson, & Anisman, 2010). These ghrelin changes were reminiscent of the ghrelin patterns observed among binge eaters, or obese individuals (Geliebter, Gluck, & Hashim, 2005; Geliebter et al., 2004; English, Ghatei, Malik, Bloom, & Wilding, 2002). However, in this study (Raspopow et al., 2010) food was offered to all participants, thus making it difficult to determine whether or not eating itself influenced the outcomes. Thus in the present investigation, we included a condition where food was or was not offered in order to determine whether the altered pattern of ghrelin observed among emotional eaters was, in fact, a function of food presentation and intake, or whether it reflected more general alterations of ghrelin among emotional eaters.

As in the case of corticosterone, stressors consistently increase ghrelin levels in rodents (Asakawa et al., 2001; Kristenssson et al., 2006; Lutter et al., 2008; Patterson, Ducharme, Anisman, & Abizaid, 2010), and in humans that showed a high cortisol response to a stressor (Rouach et al., 2007). In the case of cortisol, the rise of this hormone in certain situations, such as public speaking in the Trier Social Stress Test (TSST; Kirschbaum, 1993), may be tied to the social evaluative threat and the resulting emotions (e.g., shame) associated with this stressor (Dickerson & Kemeny, 2004; Dickerson, Mycek, & Zaldivar, 2008). However, the *anticipation* of a stressor was only associated with moderately elevated cortisol levels (Kelly, Matheson, Martinez, Merali, & Anisman, 2007). It is uncertain whether the anticipation of a stressor would affect ghrelin levels in the same way that actually experiencing the stressor would.

The primary purpose of the present study was to determine whether anticipation of a stressor would instigate food intake (when food was offered), as well as variations of plasma ghrelin and cortisol levels in humans. It was also of interest to examine whether these hormone changes would be related to altered expression of hunger or food consumption among individuals who were emotional vs. non-emotional eaters. Typically, food intake and hormone levels have been assessed after exposure to a stressor, even though *anticipation* of a stressor elicits anxiety and cortisol responses, and could potentially promote eating among emotional eaters. Thus, in the present investigation we focused on whether anticipation of a stressor was related to ghrelin and cortisol levels, and to what extent these changes accompanied particular mood responses that have been associated with cortisol changes in the TSST (e.g., shame, anger, anxiety). Specifically, it was hypothesized that cortisol and ghrelin levels would increase in anticipation of the stressor, compared to a control task, and that this effect would be exaggerated among emotional eaters. Additionally, it was expected that emotional eaters would have lower levels of basal ghrelin than non-emotional eaters, and that ghrelin levels would not decrease among emotional eaters following food intake. Finally, it was of interest to examine, the association between food intake and stressor-elicited changes in cortisol and ghrelin levels, and whether these associations would differ between emotional and non-emotional eaters.

Material and methods

Participants

Undergraduate women ($N = 103$) were recruited via an online study recruitment system for a two-part study allegedly examining student responses to an employment task (see Table 1 for distribution of participant age and ethnicity across study conditions). Exclusionary criteria for the blood sampling session included medical conditions or medications that could affect hormone release (assessed in background information questionnaires during the introduction section), as well as extreme fear of needles or previous bad experiences (e.g., nausea, fainting) while providing blood samples, which was assessed by the researcher at the end of the introduction session.

Measures

Emotional eating behavior

The Emotional Eating subscale of the Dutch Eating Behaviors Questionnaire (van Strien et al., 1986) assessed women's emotional eating behaviors. This 13-item scale uses a 5-point Likert scale ranging from 0 "Never" to 4 "Very Often" to rate the degree to which individuals have a desire to eat in response to diffuse and specific negative emotions (e.g., upset, irritated, bored, cross). This scale demonstrated excellent reliability (Cronbach's $\alpha = .96$).

Table 1
Participant Mean(SD) Age and Ethnicity as a Function of Emotional Eating Group and Condition Allocation.

	Total	Emotional eating		Stressor anticipation condition		Food condition	
		Low	High	Control	Stressor	No food	Food
Age	20.72(2.43)	20.79(5.44)	20.65(4.21)	20.89(4.92)	20.62(4.79)	21.02(4.94)	20.47(4.74)
Ethnicity							
Caucasian (%)	51.0	56.3	46.3	56.5	46.4	48.9	53.6
Middle Eastern (%)	13.7	8.3	18.5	10.9	16.1	11.1	16.1
East Asian (%)	11.8	10.4	13.0	10.9	12.5	8.9	12.5
Black (%)	11.8	12.5	11.1	8.7	14.3	11.1	12.5
Hispanic (%)	2.9	4.2	1.9	2.2	3.6	4.4	1.8
Asian (%)	2.9	4.2	1.9	2.2	3.6	4.4	1.8
Aboriginal (%)	2.0	2.1	1.9	2.2	1.8	4.4	0
Other (%)	3.9	2.1	5.6	6.5	1.8	6.7	1.8

No significant differences were observed between condition allocations for age or ethnicity in either emotional eating, stressor anticipation condition or food condition.